U.S. Department of Labor

Office of Administrative Law Judges
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In the Matter of

Gary L. Looney,
Claimant

v.

Harman Mining Company and Old Republic Insurance Company,
Employer/Carrier

and

Director, Office of Workers' Compensation Programs,
Party-In-Interest

Date Issued: June 8, 2000

Case No. 1994-BLA-433

**DECISION AND ORDER ON REMAND**

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

I have based my analysis on the entire record, including the exhibits and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.

## I. Statement of the Case

### a. Procedural History

This claim has a long procedural history. The Claimant first filed a claim for benefits on

February 1, 1993. On September 27, 1994, Administrative Law Judge Ralph A. Romano awarded benefits to the Claimant. On appeal by the Employer, the Benefits Review Board ("the Board") vacated Judge Romano's findings that the Claimant had established the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a)(4), and that the medical opinion evidence was sufficient to establish total disability due to pneumoconiosis under 20 C.F.R. § 718.204(c)(4) and (b), and remanded the claim for further consideration.[1]

In his Decision and Order on Remand dated January 29, 1996, Judge Romano again granted benefits to the Claimant. This decision was appealed to the Board, which again vacated his findings regarding 20 CFR §§ 718.202(a)(4) and 718.204(b) and (c). In his January 10, 1997, Decision and Order on Remand, Judge Romano again granted benefits; the Employer appealed. The Board affirmed Judge Romano's finding of total disability under § 718.204(c), but vacated his findings under §§ 718.202(a)(4) and 718.204(b).[2] The Board instructed Judge Romano on remand to evaluate the opinions of Drs. Sargent and Fino in light of the decision in *Warth v. Southern Ohio Co.*, 60 F.3d 173 (4th Cir. 1995), and to reconsider the opinions of Drs. Sargent, Fino, Forehand, and Robinette under §§ 718.202(a)(4) and 718.204(b).

In his July 31, 1998, Decision and Order on Remand, Judge Romano again granted the Claimant benefits. The Employer again appealed, challenging Judge Romano's weighing of the medical opinions of Drs. Sargent, Fino, Forehand, and Robinette. The Board concluded that Judge Romano did not adequately and properly weigh the opinions of these physicians under §§ 718.202(a)(4) and 718.204(b).[3]

Finally, the Board granted the Employer's request that this claim be remanded and reassigned to a different administrative law judge.

This matter was subsequently assigned to me. Pursuant to my Order of March 10, 2000, the parties were allowed to submit briefs on the issues presented on remand. On April 24, 2000, the Employer filed its brief; on June 6, 2000, the Claimant filed his brief. I have reviewed the entire exhibit record, as well as the briefs filed by the parties, in making my determination as to whether the Claimant has pneumoconiosis pursuant to § 718.202(a)(4), and whether the Claimant's total disability is due to his pneumoconiosis, under § 718.204(b).

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[1] The Board upheld Judge Romano's finding that the Claimant had sixteen and three quarter years of coal mine employment.

[2] The Board found that the opinions of Drs. Robinette and Forehand, on which Judge Romano relied, were documented and reasoned, and could, if properly credited, support an award of benefits.

[3] The Board affirmed Judge Romano's rejection of Dr. Sutherland's opinion as unreliable, because his report failed to address the Claimant's smoking history, as it was not challenged on appeal.

**b. Summary of the Medical Opinions**

The medical opinion evidence that is at issue is summarized below.[4]

*Dr. Emory Robinette*

Dr. Robinette examined the Claimant in March 1991 upon referral by Dr. Sutherland for evaluation of a syncopal episode (EX 7).[5] The Claimant reported to Dr. Robinette that he had repeated PND episodes, orthopnea, congestion, chronic cough, and dyspnea induced by exertion with "apparent near syncope." The Claimant told Dr. Robinette that in January 1991, he had "generalized anterior chest pain and headache" with an at-home syncopal episode. As a result, he had been referred to Roanoke, where he was diagnosed with primary lung problems. Dr. Robinette noted an exercise test done by Dr. Patel, who found "near syncope secondary to exertional dyspnea and breathlessness" after one minute and 27 seconds in stage I. There was no chest pain, and no cardiac disease was found based on this test. Dr. Robinette reported that the Claimant was taking both prednisone and proventil for shortness of breath. He also noted that the Claimant had a 20 to 23 year history of smoking, though he had reduced the rate of smoking from between 1.5 and 2 packs a day, to 4 cigarettes a day. He also noted that the Claimant had stopped work in February 1991, after having worked in the mines for more than 20 years, primarily as a roof bolter.

Upon examination, Dr. Robinette found the Claimant to be obese. Auscultation of the chest showed "diffuse sonorous wheezes and rhonchi in all lung fields with moderate prolongation of the expiratory phase." He reviewed an x-ray of the Claimant's chest and found "evidence of mild interstitial pulmonary fibrosis" as well as "scattered opacities . . . consistent with pneumoconiosis with a primary opacity size of P and a secondary opacity size of T with a profusion abnormality of 1/0 . . . . localized to the mid and lower lung zone." He also noted a few calcified granuloma.

Dr. Robinette had pulmonary function studies performed, which showed a decreased flow rate and FVC, as well as a diminished FEF 25-75, without improvement upon administration of bronchodilators. Lung capacity and residual volume were elevated. ABG testing showed an elevated carboxyhemoglobin level, but normal pH, PCO2, and PO2, which Dr. Robinette determined to be consistent with moderate obstructive lung disease. The air trapping was consistent with air flow obstruction.

Based on these findings, Dr. Robinette made the following diagnoses:

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[4] I have not considered Dr. Sutherland's opinions, as the Board specifically affirmed Judge Romano's rejection of them.

[5] There is no curriculum vitae for Dr. Robinette in the record; his letterhead indicates that he specializes in pulmonary medicine.

1. Simple coal workers' pneumoconiosis with a profusion abnormality of 1/0.
2. Moderate obstructive lung disease, most likely asthma.
3. History of chronic cigarette abuse.
4. Exogenous obesity.

Dr. Robinette stated that the Claimant has significant obstructive lung disease, which is so severe that he would have difficulty performing his work as an underground coal miner, particularly a roof bolter. Dr. Robinette believed the etiology of the Claimant's pulmonary disease was "probably a combination of asthma, obstructive lung disease and coal workers' pneumoconiosis." Dr. Robinette recommended that the Claimant continue his medications, quit smoking, and lose weight.

Included with Dr. Robinette's letter were laboratory reports and records from the Claimant's pulmonary evaluation, which included pulmonary function studies and arterial blood gas studies.

*Dr. J. Randolph Forehand*

Dr. Forehand examined the Claimant at the request of the Department of Labor on February 19, 1993 (DX 15).[6] The Medical History and Examination Form that Dr. Forehand completed reflects that the Claimant was a miner for 25 years, 21 years of that underground. It also reflects that the Claimant began smoking a quarter pack of cigarettes daily beginning in 1978. The Claimant reported wheezing, coughing, dyspnea, and orthopnea, as well as black-out spells and shortness of breath. Upon examination of the Claimant, Dr. Forehand detected wheezing in the right chest posteriorly, and post nasal drip. Dr. Forehand concluded from the x-ray he ordered that the Claimant had interstitial scarring. The Claimant's pulmonary function study showed an obstructive ventilatory pattern, and his arterial blood gas study showed hypoxemia, both at rest and with exercise. His EKG was normal. Dr. Forehand diagnosed the Claimant with chronic airflow obstruction, based on his history and physical examination, and the results of his pulmonary function and arterial blood gas studies. In his opinion, the etiology of this illness was coal dust exposure, cigarette smoking, and airway hyperactivity. Dr. Forehand felt that the combination of the Claimant's airflow obstruction and arterial hypoxemia made him unable to perform his last coal mining work. In Dr. Forehand's opinion, the Claimant's disability was due to a combination of his smoking and his coal dust exposure.

Dr. Forehand was subsequently asked by the Department of Labor to respond to specific questions about the Claimant's condition (DX 16). In a letter dated April 16, 1993, Dr. Forehand reported that he had reviewed the Claimant's black lung evaluation, and concluded that he has coal workers' pneumoconiosis (DX 17). He indicated that he was using the legal, and not the medical definition of coal workers' pneumoconiosis, and was not relying exclusively on the appearance of the x-

[6] There is no curriculum vitae in the record for Dr. Forehand. His letter of April 16, 1993 reflects that he is a B reader.

ray. Dr. Forehand pointed out that coal workers' pneumoconiosis can arise in the absence of changes on a chest x-ray. In his opinion, the Claimant is totally disabled, and could not return to his last coal mine employment; this disability is due at least in part to his coal mine employment.

*Dr. Gregory J. Fino*

Dr. Fino, who is board-certified in Internal Medicine and Pulmonary Disease and is a B-reader (EX 12), reviewed medical records at the request of the Employer, and prepared a report dated June 14, 1994 (EX 9). In reviewing Dr. Robinette's 1991 report, Dr. Fino concluded that the spirometry results obtained by Dr. Robinette were invalid, due to premature termination of exhalation, and a lack of reproducibility in the expiratory tracings, as well as a lack of abrupt onset to exhalation. Dr. Fino felt that the values represent the Claimant's minimal lung function, but not his maximum lung function. In addition, Dr. Fino concluded that the MVV results were invalid, because the individual breath volumes were shallow and less than 50% of the FVV, with erratic individual breath volumes. He noted that the breathing frequency was less than 60 breaths per minute. In his opinion, the MVV value underestimated the Claimant's true lung function, and was not evidence of respiratory impairment.[7] Dr. Fino noted that after the administration of bronchodilators, there was a better effort on the FVC, which was in the normal range, but he still felt that the Claimant did not give maximum effort. He noted that the Claimant's lung volumes were increased, and his diffusing capacity was normal.

Dr. Fino noted that the Claimant was being treated with bronchodilator medication, which is not the mode of treatment for pneumoconiosis, as it works in reversible lung disease. Coal mine dust does not cause a reversible narrowing of the breathing tubes, but causes abnormality in the lungs which does not improve with the use of bronchodilators.[8]

Dr. Fino also reviewed the results of the DOL examination conducted by Dr. Forehand on February 19, 1993. He noted that the Claimant reported a shorter period of smoking, as well as a lighter amount, to Dr. Forehand than what he had told Dr. Robinette. Dr. Fino noted that the spirometry results showed moderate obstruction, with elevated lung volumes. The arterial blood gas studies showed hypoxia at rest and exercise, which Dr. Fino felt represented the Claimant's lung disease due to smoking. Dr. Fino stated that there was no worsening of the oxygenation after exercise, as would be expected with a coal mine dust related condition.

Dr. Fino also reviewed Dr. Forehand's letter of April 16, 1993, in which Dr. Forehand stated that the Claimant meets the legal definition of pneumoconiosis, but not the medical definition. Dr. Fino felt

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[7] Dr. Fino cited to three medical references on spirometry.

[8] Dr. Fino noted that "there is no good clinical evidence in the medical literature that coal mine dust inhalation in and of itself causes significant obstructive lung disease irrespective of its ability to be reversed following bronchodilators."

that, even using the legal definition, the Claimant's abnormality is not consistent with a coal mine dust-induced condition, because it is a pure obstructive abnormality with elevation in the lung volumes. Dr. Fino agreed with Dr. Forehand that a chest x-ray cannot absolutely exclude a diagnosis of pneumoconiosis. However, Dr. Fino felt that other information, such as spirometry, lung volumes, and diffusing capacity, can be used to determine the presence or absence of pneumoconiosis. In this case, he felt that there was none.

Dr. Fino reviewed the report of Dr. Sargent dated September 7, 1993. He first noted that the Claimant reported less of a smoking history than he had to Dr. Robinette. Dr. Fino noted the results of the lung examination, which showed a prolongation of the expiratory phase; and the pulmonary function tests, which showed a moderate obstructive abnormality. He also noted that the lung volumes were elevated, with a normal diffusing capacity.

Based on a review of all of the medical evidence, Dr. Fino concluded that the Claimant does not have an occupationally acquired pulmonary condition due to coal mine dust exposure. Dr. Fino based this conclusion on the fact that the majority of the x-ray readings are negative, as are his readings of two of the x-rays. In addition, the spirometric evaluations show a purely obstructive ventilatory abnormality, without a restrictive defect; there is also obstruction in the small airways. Dr. Fino pointed out that the Claimant's small airway flow was more reduced, on a proportional basis, than the large airway flow, a finding that is not consistent with a coal dust related condition, but that is consistent with conditions such as cigarette smoking, emphysema, non-occupational chronic bronchitis, and asthma.

Dr. Fino also noted that the Claimant improved after the use of bronchodilators, as shown on two of the pulmonary function studies. This implies that the cause of the obstruction is not fixed and permanent. Pneumoconiosis, however, is a fixed condition, and thus bronchodilators would be of no benefit. Therefore, improvement following bronchodilators, or reversibility, is clearly evidence that a non-occupationally acquired pulmonary condition is causing the obstruction.

Dr. Fino stated that the Claimant has elevated lung volumes, a condition that is due to obstructive lung disease. In contrast, under-inflated conditions are due to the contraction of fibrotic scarring, as seen in pulmonary fibrosis. The air-trapping pattern is typical to patients with obstructive lung diseases such as emphysema, asthma, or chronic obstructive bronchitis, or a combination thereof. But it is not a pattern that is consistent with the contraction of lung tissue due to fibrosis, as would be expected with pneumoconiosis. In Dr. Fino's opinion, the Claimant's normal diffusing capacity values rule out the presence of clinically significant pulmonary fibrosis, of which pneumoconiosis is an example. Dr. Fino pointed out that the Claimant has hypoxia at rest and with exercise, with no worsening; in light of his normal diffusing capacity, the hypoxia cannot be attributed to lung destruction due to fibrosis. Rather, his hypoxia is due to a ventilation perfusion abnormality, caused by his cigarette smoking.

Dr. Fino stated that there is no evidence of any interstitial pulmonary condition, as would be caused by pneumoconiosis. He stated that a respiratory impairment due to interstitial pulmonary

conditions is different from that present in an obstructive condition, and he enumerated examples of the type of abnormalities that can occur in interstitial pulmonary conditions. They include a reduction of the forced vital capacity in the absence of obstruction; a significant reduction in the diffusing capacity in the absence of anatomic emphysema; a drop in the p02 with exercise in the absence of other etiologies; or reduced lung volumes in the absence of other etiologies. Noting that pneumoconiosis is an interstitial pulmonary condition, Dr. Fino concluded that the information from the Claimant's medical records shows a pure obstructive ventilatory abnormality with no evidence of interstitial disease.

Noting Dr. Forehand's statement that the legislative definition of pneumoconiosis would allow a person with obstruction such as the Claimant's to establish the existence of pneumoconiosis, Dr. Fino stated:

> The question arises, however, whether pneumoconiosis can cause an obstructive abnormality or emphysema. It is my opinion that it cannot based on the following review of the medical literature.

Dr. Fino proceeded to discuss medical literature establishing a distinction between pathological emphysema, which can be caused by coal dust, and which is described as focal emphysema, and clinical emphysema, which is caused by cigarette smoking, and is known as centrilobular emphysema.[9] Dr. Fino reviewed several studies discussing this distinction, and discounted studies suggesting a connection between clinical emphysema and coal miners.[10] In particular, he referred to Dr. Morgan's *Occupational Lung Diseases* (1984), noting that coal workers' pneumoconiosis is associated with focal emphysema, with no clinical correlate. Dr. Fino stated:

> Dr. Morgan states that the changes of focal emphysema "cannot be equated with airway obstruction and it has been repeatedly demonstrated that an increasing category of simple coal workers' pneumoconiosis, although associated with increasing focal emphysema, does not lead to any concomitant increase in airway obstruction." Of course, airway obstruction is what is necessary to make emphysema "clinically significant."

Dr. Fino noted that the U.S. Surgeon General's 1985 report found that the death rate from emphysema and bronchitis was not increased in coal miners and the mortality rate did not go up with exposure to dusty conditions. According to Dr. Fino, the 1985 report also found that coal dust inhalation did not contribute to excessive morbidity or mortality for lung conditions such as simple CWP,

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[9] Dr. Fino also referred to another type of cigarette smoking-induced emphysema, called panacinar emphysema, which has never been attributed to coal workers' pneumoconiosis.

[10] Dr. Fino opined that studies finding otherwise were flawed, and he provided examples of these flaws, which included inadequate controls for smoking and inadequate numbers of non-smoking miners used in the studies.

emphysema, asthma, tuberculosis, or pneumonia, but rather only increased in progressive massive fibrosis. Dr. Fino quoted language from the Surgeon General's report which suggested that emphysema in coal miners was focal emphysema and did not increase morbidity or mortality in coal miners. Dr. Fino also distinguished the British studies finding clinical emphysema in coal miners, based on inadequate controls and differences in the rank of coal involved. He discredited the opinion of Dr. Ruckley that coal dust exposure can cause significant obstructive impairment, based on the lack of a standard method of lung inflation, abnormal methodology of the pulmonary function studies, and small sample size. He also took issue with her determination that centrilobular emphysema arises from coal dust exposure, citing the overly broad definition of centriacinar emphysema employed by Dr. Ruckley.

Dr. Fino reviewed several studies finding that coal mine dust inhalation caused an obstructive ventilatory abnormality, and discounted them, noting, *inter alia*, that they did not discuss a relationship between the FEV1/FVC ratio and coal mine dust inhalation, and that obstruction is only diagnosable by a reduction in the FEV1/FVC. Dr. Fino stated:

> If there is no relationship between coal mine dust inhalation and the FEV1/FVC, then there can be no association between coal mine dust inhalation and obstruction.

Dr. Fino cited to several studies, including one by Dr. Rasmussen involving southwest Virginia coal miners, and concluded that:

> This information clearly shows that obstruction is not a clinically significant abnormality arising out of the inhalation of coal mine dust.

Dr. Fino also cited to a study by Drs. Cooper and Johnson, *Exercise Capacity and Coal Workers' Pneumoconiosis: An Analysis Using Causal Modelling,* British Journal of Industrial Medicine (1990), noting that:

> Their conclusions noted that coal mine dust exposure irrespective of the chest x ray findings did not affect the FEV1. That is simply to state that the FEV1 was not decreased as a result of coal mine dust inhalation. What was decreased were forced vital capacity and exercise capacity which are the classic findings one would expect in an interstitial pulmonary condition. Hence, Drs. Cooper and Johnson provide further information clearly showing that there is not an obstructive ventilatory abnormality arising out of coal mine dust exposure as has been suggested by authors publishing in the British literature.

He felt that the literature indicated that clinical emphysema must show a clinically significant obstruction as evidenced by "a reduction in the FEV1/FVC ratio in conjunction with a reduction in the FEV1 and a reduced diffusing capacity." He stated that such a reduction has not been seen in non-smoking miners, and that lung function decreases with age at a rate of 20-30 cc per year, as opposed to a 9 cc per year rate of decrease attributed to pneumoconiosis by Attfield. Dr. Fino discounted Attfield and

Hodous' finding that inhalation of coal dust caused obstructive ventilatory impairment, because they did not find a relationship between coal dust inhalation and the FEV1/FVC ratio. Dr. Fino stated: "Clearly, obstruction is only diagnosable by a reduction in the FEV1/FVC." Further, Attfield and Hodous' 1992 study of the FEV1/FVC ratio included anthracite miners, who were distinguishable from coal miners, and did not find a FEV1 decrease that was significant, compared with the decrease that occurs as a result of aging.

Dr. Fino interpreted Dr. Rasmussen's research as showing that "obstruction is not a clinically significant abnormality arising out of coal mine dust." Drs. Cooper and Johnson's research showed that FEV1's did not increase regardless of what was shown on the chest x-rays. However, FVC's and exercise capacities did decrease, as is consistent with interstitial pulmonary conditions. Dr. Fino discounted the findings of the National Coal Board of Great Britain that showed increased obstruction in miners because the studies did not adequately control for cigarette smoking when the study began. Further, the performing and interpreting of the pulmonary function studies were flawed, and obstruction was defined differently than it was in the United States. From his review of the literature, Dr. Fino concluded that "there is no evidence of an increase in disabling or impairing emphysema in coal miners. Although emphysema is a pathological entity seen in coal workers' pneumoconiosis, coal workers' pneumoconiosis does not cause clinical emphysema."

In conclusion, Dr. Fino found that there was insufficient objective medical evidence to justify a diagnosis of simple coal workers' pneumoconiosis. In his opinion, the Claimant does not suffer from an occupationally acquired pulmonary condition, although he has a disabling respiratory impairment due to cigarette smoking. Dr. Fino stated that the Claimant would be as disabled if he had never set foot in the coal mines.

Dr. Fino gave testimony by deposition on June 22, 1994 (EX 14). He noted that since he had prepared his report, he had reviewed four additional chest x-rays of the Claimant. Based on the evidence detailed in his report as well as the additional x-rays, he concluded that the Claimant does not have coal workers' pneumoconiosis, but that he does have a pulmonary impairment due to his history of cigarette smoking. Dr. Fino noted that a diagnosis of pneumoconiosis is based on clinical, objective medical information. While the symptoms and history of a patient, as well as findings on physical examination, may be relevant to determining whether a person has a respiratory impairment, they are not helpful in making a diagnosis as to the existence of pneumoconiosis, because many conditions can cause the same symptoms and clinical findings as pneumoconiosis. Rather, Dr. Fino relies on objective tests, including x-rays and pulmonary function testing, to determine if the results are consistent with pneumoconiosis or some other lung condition. Dr. Fino did note that there are physical examination findings that are consistent with pneumoconiosis, such as rales in the lungs due to scarring or fibrosis; however, wheezes or rhonchi are not found in coal workers' pneumoconiosis. Dr. Fino noted that the Claimant's exposure to coal mine dust would be sufficient to cause pneumoconiosis in a susceptible individual. In addition, his exposure to cigarette smoking was also sufficient to cause a cigarette smoking-induced lung condition, if he were a susceptible individual.

Dr. Fino found no pattern of pneumoconiosis on any of the x-ray films he reviewed. The spirograms conducted on the Claimant showed a normal FVC, both before and after medication, unlike the reduction that would be expected in a person with pneumoconiosis. The spirograms did show significant obstruction, which is "not the type of functional abnormality that is seen in coal mine dust-related conditions, but is the characteristic type of abnormality that is seen in cigarette smoking-induced lung problems" (EX 14 at p.21). Dr. Fino explained that cigarette smoking typically affects the small airways in greater proportion than the large airways; the Claimant's spirograms show a more proportional reduction in the small airway flow, as measured by the FEF 25-75, than the large airway flow, as measured by the FEV1. In addition, the Claimant's lung volume measurements were elevated, which is consistent with cigarette smoking as an etiology for his underlying obstructive condition, rather than pulmonary fibrosis (of which pneumoconiosis is an example), which produces under-inflation, and low or decreased lung volume measurements. Dr. Fino found no evidence in the lung volume studies of the presence of a restrictive disease.

Dr. Fino also noted that the Claimant's diffusing capacity was normal, indicating that there is no lung destruction, and that pneumoconiosis is not present. He noted that two conditions can result in a reduction of diffusion capacity by causing lung destruction: emphysema and pulmonary fibrosis.

Dr. Fino also discussed the variability in the blood gas testing results, noting that it implied the absence of a fixed irreversible condition, such as pneumoconiosis. In contrast, cigarette smoking-induced conditions can cause variability in the blood gas value. He also noted that the p02 values at rest and exercise, which showed variable hypoxia, were consistent with cigarette smoking, but not pneumoconiosis, which would cause chronic hypoxia.

Dr. Fino did not agree with Dr. Forehand's conclusion that the Claimant has legal pneumoconiosis. He stated that pneumoconiosis does not cause pure obstruction, and that the Claimant has obstruction without any restriction or interstitial lung disease. Dr. Fino believed that the Claimant has a smoking-related condition, but not a coal mine dust-related condition. In considering whether smoking-related conditions are contributed to or aggravated by the inhalation of coal mine dust, he noted that there were no studies finding that the two are multiplicative or synergistic. He stated that while a person can have both smoking disease and coal mine dust-induced disease, one does not make the other worse. Dr. Fino stated that in the Claimant's case, he did not find any evidence, by experience, review of the medical literature (which he did not specify), or review of the file, that coal mine dust inhalation contributed to, caused, aggravated or exacerbated the Claimant's underlying nonoccupational condition. He noted that conditions other than fibrotic scarring, which may be associated with coal mine dust inhalation, primarily occur in working miners, and dissipate after a miner leaves the mines.

*Dr. Jeffrey Dale Sargent*

Dr. Sargent, who is board-certified in Internal Medicine, Pulmonary Disease, and Critical Care and is a B-reader, examined the Claimant on September 7, 1993, at the request of the Employer, and

prepared two reports. In his first report, dated September 7, 1993, Dr. Sargent indicated that he had performed an electrocardiogram, an arterial blood gas study, a carboxyhemoglobin level test, a pulmonary function test, and a chest x-ray (DX 38). He concluded that the x-ray was negative for pneumoconiosis, that the carboxyhemoglobin level indicated smoking in excess of the Claimant's claimed smoking habit of 1 pack every 3 days, and that the pulmonary function test indicated a

> moderate obstructive impairment with air trapping, hyperventilation, and normal diffusion consistent with a combination of chronic bronchitis and possible asthma, although strict criteria for reversible airways obstruction were not met on these pulmonary functions.

Dr. Sargent concluded that the Claimant does not have coal worker's pneumoconiosis, based on his negative x-ray and the "character of his ventilatory impairment." Dr. Sargent stated that pneumoconiosis causes an impairment in the presence of a positive x-ray, an impairment in a mixed obstructive and restrictive pattern. The Claimant's x-ray was not positive, and his impairment was obstructive, without evidence of restriction. He noted that cigarette smoking has been shown to cause a purely obstructive impairment of the type suffered by the Claimant, and he therefore concluded that the minimal interstitial changes shown on the Claimant's x-ray, and the obstructive nature of his impairment, are perfectly consistent with impairment due to his previous and ongoing smoking habit. Assuming that the Claimant's previous job as a roof bolter required heavy manual labor, Dr. Sargent felt that the Claimant would have difficulty performing that job. Dr. Sargent concluded that the Claimant has a moderate obstructive ventilatory impairment due to cigarette smoking, but not due to coal dust exposure. Dr. Sargent felt that the Claimant's condition might improve with the cessation of cigarette smoking and a more aggressive bronchodilator regimen.

At the Employer's request, Dr. Sargent reviewed the Claimant's medical records and prepared a report dated May 26, 1994, addressing specific questions posed by Employer's then-counsel (EX 8). Dr. Sargent was asked whether the Claimant has pneumoconiosis, defined as "a chronic dust disease of the lung or sequelae thereof, including respiratory or pulmonary impairments, due in whole or in part to, significantly related to or substantially aggravated by dust exposure or coal mine employment." In Dr. Sargent's opinion, the Claimant does not have pneumoconiosis, because only one x-ray was read as positive for pneumoconiosis, and

> [t]he medical literature suggests that the chest x-ray interpretation is the most sensitive indicator of dust burden of the lung. Therefore, I would conclude, based on this data, that it would be very difficult to diagnose coal worker's pneumoconiosis. Also, although there are pulmonary function abnormalities, these are not characteristic of the type caused by coal worker's pneumoconiosis.

Dr. Sargent characterized the Claimant's impairment as a "moderately obstructive partially reversible ventilatory impairment." He noted that Dr. Forehand had found partial reversibility, while his own studies and those of Dr. Robinette did not show reversibility. Dr. Sargent again opined that the Claimant's condition prevented him from performing his work as a roof bolter; however, this impairment

was not related to his coal mining work, but rather was related to his history of cigarette smoking. Dr. Sargent reasoned as follows:

> [The Claimant] has two pulmonary exposures that put him at risk for development of lung disease, those being cigarette smoking and coal dust exposure. These two causes of impairment are distinguishable from one another on the basis of objective criteria. Coal worker's pneumoconiosis, when it causes impairment, does so in the face of a positive x-ray, which clearly is not present in this case. It also causes a mixed obstructive and restrictive pattern which is irreversible with bronchodilator. Cigarette smoking, on the other hand, causes a purely obstructive impairment without restriction, which can be reversible with bronchodilator, and may cause impairment in the face of an x-ray negative for characteristic changes of pneumoconiosis. Therefore, all of the objective evidence in this case supports the cause of this impairment being cigarette smoking and not coal dust exposure. He has a partially reversible, purely obstructive impairment with a negative x-ray.

While Dr. Sargent felt that the Claimant could not presently perform his usual coal mine work, he reiterated his earlier statement that he was unsure whether such impairment was permanent in nature, and might not improve if the Claimant stopped smoking and were put on a more aggressive bronchodilator regimen.

Dr. Sargent testified by deposition on July 5, 1994 (EX 13). Dr. Sargent noted the Claimant's 20 year history of coal mine employment, as well as his history of cigarette smoking, both of which he described as the Claimant's two pulmonary risk factors. While Dr. Sargent considered the Claimant's history of coal mine employment to be sufficient, if he were a susceptible host, to contract pneumoconiosis, he also considered his history of smoking to be sufficient to cause respiratory disability if he were a susceptible host. Dr. Sargent noted that the x-ray he took was negative for pneumoconiosis, because although there were opacities, they were irregular instead of rounded, as would be the case if they were caused by coal dust, and they were located at the base of the lung rather than the apex, the location favored for coal dust related opacities. Dr. Sargent stated that the pulmonary function tests he administered resulted in normal forced vital capacity results. However, the FEV1 value was only 61%, resulting in a ratio of FEV1 to FVC that was lower than normal. According to Dr. Sargent, this is consistent with moderate obstruction. There was no significant improvement with the use of bronchodilators. He also noted evidence of hyperinflation and air trapping, both of which are consistent with obstructive lung disease. The Claimant's diffusing capacity was normal. The Claimant's arterial blood gas studies showed mild hypoxemia, as well as an elevated carboxyhemoglobin level, which was much higher than would be expected in a person who smoked a third of a pack of cigarettes a day, as the Claimant stated. According to Dr. Sargent, the Claimant's carboxyhemoglobin level was consistent with a pack and a half to two packs a day. Dr. Sargent noted the results obtained by Dr. Forehand in February 1993, which were not as good as the results obtained by Dr. Sargent; he stated that although this variability in results over time is consistent with obstructive disease, it is not consistent with coal workers' pneumoconiosis, which is irreversible, and would not result in blood gas abnormalities that wax

and wane over time.

Dr. Sargent concluded that the Claimant's respiratory impairment is purely obstructive, moderate in severity. He noted that although there was no improvement with bronchodilators in his pulmonary function studies, other studies he reviewed showed some reversibility, which is not consistent with coal workers' pneumoconiosis. In Dr. Sargent's opinion, the Claimant's total respiratory disability is due to cigarette smoking, and he would be just as disabled if he had never set foot in a coal mine.

## DISCUSSION

In its Decision and Order on Remand, the Board concluded that Administrative Law Judge Romano had not weighed the relative merits of the opinions of Dr. Forehand and Dr. Robinette as against the conflicting opinions of Dr. Sargent and Dr. Fino, with respect to the existence of pneumoconiosis, as well as the etiology of the Claimant's total respiratory disability. In addition, the Board also found that Administrative Law Judge Romano erred in interpreting Dr. Fino's testimony as hostile to the Act, noting that

> Nothing in Dr. Fino's opinion or testimony indicates that he believes pneumoconiosis cannot contribute to an obstructive impairment, or that, as a rule, pneumoconiosis never causes obstructive lung disease. Dr. Fino's testimony does not go that far. Dr. Fino testified that pneumoconiosis does not cause *pure* obstruction, and this testimony is consistent with his indication in his consultative report that one would expect a restrictive impairment with pneumoconiosis.

Decision and Order at 6.

I note at the outset that, as pointed out by the Board, both Dr. Sargent and Dr. Fino have credentials that are superior to those of Dr. Robinette and Dr. Forehand. In addition, both Dr. Sargent and Dr. Fino have offered much more in the way of supporting rationale for their conclusions than Dr. Robinette and Dr. Forehand. But there are aspects of their opinions that are troubling, and that give me pause in weighing the relative merits of the physicians' opinions, as directed by the Board.

The Board rejected Judge Romano's findings that Dr. Fino's opinions were hostile to the Act. The discussion of this issue by Judge Romano in his July 31, 1998 Decision and Order on Remand refers only to Dr. Fino's deposition testimony, that "[p]neumoconiosis does not cause pure obstruction."

My review of Dr. Fino's statements, as reflected in his report and his deposition testimony, convinces me that Dr. Fino does indeed believe that pneumoconiosis does not cause obstructive lung disease. Thus, I respectfully disagree with the Board, and conclude that Dr. Fino does in fact "go that far."

Dr. Fino consistently based his conclusions that the Claimant does not have pneumoconiosis on the fact that the Claimant has a purely obstructive ventilatory abnormality, with no evidence of restriction or interstitial disease. In fact, in discussing Dr. Forehand's statements, that the Claimant's obstructive disease meets the legislative definition of pneumoconiosis, Dr. Fino stated:

> The question arises, however, whether pneumoconiosis can cause an obstructive abnormality or emphysema. It is my opinion that it cannot based on the following review of the medical literature.

Dr. Fino proceeded with an exhaustive discussion discounting studies showing that coal mine dust inhalation can cause an obstructive ventilatory abnormality. While Dr. Fino conceded that coal mine dust inhalation can cause "pathological" emphysema, a phenomenon that does not cause impairment, he concluded that it cannot cause clinical emphysema, which is an obstructive impairment. According to Dr. Fino, because the emphysema (and hence obstruction) that can be caused by coal mine dust inhalation is not clinically significant, and does not result in objective signs of impairment, it does not qualify as legal pneumoconiosis.

The reasonable inference to be drawn from Dr. Fino's extended discussion is that he believes that coal mine dust inhalation does not cause any obstructive condition that results in functional abnormalities; in other words, if a person has functional abnormalities which are caused by obstructive disease, that obstructive disease cannot be attributed to exposure to coal mine dust, or to pneumoconiosis. Dr. Fino was at great pains to explain that coal mine dust exposure does not result in <u>any</u> type of clinically significant obstructive disease, and thus that obstructive disease can never be considered to be pneumoconiosis, legal or otherwise. Thus, although Dr. Fino may seem to imply that pneumoconiosis would be expected to cause a mixed pattern of obstructive and restrictive disease, a careful reading of his statements in their totality clearly shows that he believes that pneumoconiosis manifests itself only in restrictive disease; and that a person who has obstructive disease, whether pathological, clinical, or both, must also have restrictive disease to be diagnosed with pneumoconiosis. In other words, Dr. Fino does not accept the concept of "legal pneumoconiosis," a concept firmly established by the Act, and repeatedly confirmed by the Courts and the Board.

Dr. Fino went to great lengths to differentiate cigarette smoking induced lung disease from coal dust induced lung disease, based on the differing nature of the impairment that he believes they produce. Dr. Fino stated categorically his belief that pneumoconiosis does not cause clinically significant obstructive impairment; as the Claimant has only a severe obstructive impairment, Dr. Fino concluded that he does not have pneumoconiosis, since he suffers a type of impairment (chronic obstructive disease) which Dr. Fino believes pneumoconiosis cannot produce. Based on my review of Dr. Fino's report and testimony, considered in their totality, I find that Dr. Fino ruled out pneumoconiosis as a cause of the Claimant's impairment based on his belief that pneumoconiosis does not produce obstructive disorders. This is clearly contrary to the Court's decision in *Warth*, as clarified by *Stiltner*. *See also, Bradberry v. Director, OWCP*, 117 F.3d 1361 (11th Cir. 1997).

I also have difficulty with Dr. Sargent's opinions. Dr. Sargent noted that the Claimant had pulmonary function abnormalities, but that they were not "characteristic of the type caused by coal worker's pneumoconiosis." Dr. Sargent's discussion reflects that the characteristic he attributes to coal workers' pneumoconiosis is "a mixed obstructive and restrictive pattern," unlike the purely obstructive impairment caused by cigarette smoking. As the Claimant has a purely obstructive pattern of impairment, it follows that it cannot be attributed to pneumoconiosis. The logical inference to be drawn from Dr. Sargent's discussion is that he believes that pneumoconiosis can only be diagnosed if there is some restrictive disease present, and that obstructive disease, by itself, does not qualify. This, too, is contrary to the Court's decisions in *Warth* and *Stiltner*.

Additionally, a review of Dr. Sargent's statements shows that he is of the opinion that a diagnosis of pneumoconiosis can only be made in the case of a person with a clinical impairment if that person has a positive x-ray. An administrative law judge may give less credit to the opinion of a physician which is contrary to, or in conflict with, the spirit and purposes of the Act. The opinion of a physician that he or she would not diagnose pneumoconiosis in absence of a positive x-ray is hostile to the Act. *Black Diamond Coal Co. v. BRB [Raines]*, 758 F.2d 1532 (11th Cir. 1985).

I find that the views of Dr. Sargent and Dr. Fino, as discussed above, seriously diminish the weight I am willing to accord to their opinions. The Board, in its September 28, 1999 decision remanding this matter held that

> [t]he opinions of Drs. Robinette and Forehand are reasoned and documented and could, if properly credited, support an award of benefits . . . .

However, the Board determined Judge Romano did not adequately discuss the relative merits of the opinions of Drs. Robinette, Forehand, Sargent, and Fino with respect to the issue of the existence of pneumoconiosis, as well as the issue of the cause of the Claimant's total respiratory disability. I find that, given the diminished weight I am willing to accord to the opinions of Dr. Sargent and Dr. Fino, for the reasons discussed above, the opinions of Dr. Robinette and Forehand outweigh those of Dr. Fino and Dr. Sargent; and further, that the superior qualifications of Dr. Fino and Dr. Sargent are not sufficient to tip the balance back in favor of their opinions. Thus, relying on the opinions of Dr. Robinette and Dr. Forehand, I find that the Claimant has established by a preponderance of the evidence that he has pneumoconiosis pursuant to 20 C.F.R. § 718.202(a)(4), which has resulted in a totally disabling respiratory impairment under 20 C.F.R. § 204(c). As the Claimant is entitled to the presumption (which has not been rebutted) that his pneumoconiosis arose out of his coal mine employment, the Claimant has met all of the requirements of the Act, and he is entitled to benefits.

## CONCLUSION

The Claimant has established that he suffers from pneumoconiosis that arose out of his coal mine

employment, and that he is totally disabled due to pneumoconiosis. The Claimant is therefore entitled to benefits.

## ORDER

It is ordered that the claim of Gary L. Looney for benefits under the Black Lung Benefits Act is hereby GRANTED.

It is further ordered that the Employer, Harman Mining Corporation, shall pay to the Claimant all benefits to which he is entitled under the Act commencing as of February 1993.[11]

Linda S. Chapman
Administrative Law Judge

## ATTORNEY FEES

An application by claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

**NOTICE OF APPEAL RIGHTS:** Pursuant to 20 C.F.R. 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601,Washington, D.C. 20013-7601. *A copy of a Notice of Appeal must also be served on*

[11] I find that the record does not establish the date of the Claimant's disability, and thus benefits commence in the month and year in which his claim was filed.

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